



Original Article

Drug-induced sedation endoscopy in pediatric obstructive sleep apnea syndrome ☆

A. Boudewyns^{a,*}, S. Verhulst^b, M. Maris^a, V. Saldien^c, P. Van de Heyning^a^a Department of Otorhinolaryngology, Head and Neck Surgery, University Hospital Antwerp, Antwerp University, Belgium^b Department of Pediatrics, University Hospital Antwerp, Antwerp University, Belgium^c Department of Anesthesiology, University Hospital Antwerp, Antwerp University, Belgium

ARTICLE INFO

Article history:

Received 17 February 2014

Received in revised form 18 June 2014

Accepted 21 June 2014

Available online 27 August 2014

Keywords:

Upper airway

Obstructive sleep apnea

Adenotonsillectomy

Pediatric

Endoscopy

ABSTRACT

Aim: To describe the pattern of upper airway (UA) obstruction during drug-induced sedation endoscopy (DISE) and to evaluate the outcome of DISE-directed treatment.**Methods:** Prospective study of DISE in surgically naive obstructive sleep apnea syndrome (OSAS) children without syndromic comorbidity or craniofacial abnormalities. Treatment was individually tailored according to UA findings during DISE and polysomnographic data. Reported values are median (lower–upper quartile).**Results:** Thirty-seven children aged 4.1 years (2.1–6.0), with body mass index z-score 0.3 (–0.9 to 0.9), and obstructive apnea–hypopnea index (oAHI) 9.0/h (6.1–19.3) were included. Adenotonsillar obstruction was found in 33 cases (89%) as an isolated entity or as part of a multi-level obstruction. These children were treated with adenotonsillectomy ($n = 28$), adenoidectomy ($n = 3$), or tonsillectomy ($n = 2$). The remaining four patients received non-surgical treatment. Pre-postoperative polysomnographic data in 22 patients showed a significant improvement in oAHI from 8.6/h (6.7–20.7) to 1.0/h (0.6–2.0) ($P = 0.001$). Only two of these 22 children had residual OSAS (oAHI ≥ 5 /h), indicating a success rate of 91%.**Conclusions:** Based on UA findings during DISE, a non-surgical treatment was proposed for 11% of children. A 91% success rate was obtained in those treated with (adeno)tonsillectomy. These data suggest that DISE may be helpful to identify patients most likely to benefit from UA surgery.

© 2014 Elsevier B.V. All rights reserved.

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is a frequent condition in otherwise healthy children with an overall prevalence of 1% to 4% [1] and adenotonsillectomy (ATE) is the primary treatment for pediatric OSAS. Although an improvement in polysomnographic (PSG) parameters is observed in the vast majority, 13–29% of children in a relatively low-risk population have persistent OSAS defined as an apnea/hypopnea index (AHI) ≥ 5 /h [2,3].

In adults, upper airway (UA) endoscopy during sedation – also called drug-induced sedation endoscopy (DISE) – is considered a valuable tool in selecting patients for UA surgery or oral appliance therapy [4]. Recently, it was shown that the outcome of

UA surgery in adults may be at least partially determined by DISE findings [5].

The use of DISE in children with complex UA obstruction was first described in 2000 by Myatt and Beckenham [6]. Fishman et al. compared DISE with awake endoscopy in children with persistent OSAS following ATE and concluded that DISE has a better interobserver correlation for the nasopharynx and supraglottis, and identifies more cases of obstruction located at the nasopharynx, lateral pharyngeal wall/tongue base, and supraglottis [7]. Truong et al. [8] reported the reliability of pediatric DISE in a heterogeneous group of 80 patients and DISE-directed treatment outcomes in 35 children undergoing ATE. However, 28.2% of the subjects were considered hypotonic or had syndromic comorbidities [8]. Prospective studies on outcomes following DISE-directed treatment in surgically naive patients without comorbidity or craniofacial abnormalities are presently not available in the pediatric OSAS population.

Since July 2011, we have routinely performed DISE in children diagnosed with OSAS and considered candidates for UA surgery.

We conducted a prospective study with the following aims: (1) to describe the pattern of UA obstruction observed during DISE in

☆The results of this study were presented in part at the European Respiratory Society Annual Congress, Barcelona, Spain, 7–11 September, 2013; Abstract 850814.

* Corresponding author at: University Hospital Antwerp, Department of Otorhinolaryngology, Head and Neck Surgery, Wilrijkstraat 10, 2650 Edegem, Belgium. Tel.: +32 3 821 42 44; fax: +32 3 825 05 36.

E-mail address: an.boudewyns@uza.be (A. Boudewyns).

children with OSAS without previous UA surgery for snoring or OSAS, defined as surgically naive and without craniofacial anomalies or syndromes associated with OSAS; (2) to evaluate outcomes in those selected for UA surgery based upon UA findings during DISE.

2. Methods

All children meeting the inclusion criteria and scheduled for DISE between July 2011 and August 2013 were prospectively enrolled. Inclusion criteria were a diagnosis of OSAS and no previous history of adenoidectomy and/or tonsillectomy for OSAS. Children with an underlying medical condition or syndrome associated with OSAS and possibly affecting UA morphology, such as craniofacial malformations and trisomy 21, were excluded.

Diagnosis of OSAS was based upon a combination of clinical signs and symptoms of UA obstruction and full-night PSG. Tonsils were graded according to the Brodsky score [9]. Body mass index (BMI) was calculated as weight (kg)/height (m²) and body mass index (BMI) z-scores were calculated according to Flemish growth curves for boys and girls (KIGS calculator by Pfizer).

All children underwent nocturnal PSG for ≥6 h on the day of admission at the Pediatric Sleep Disorders Center of the Antwerp University Hospital, Belgium. The following variables were continuously measured and recorded by a computerized polysomnograph (Brain RT, OSG, Rumst, Belgium): electroencephalography (C4/A1 and C3/A2); electro-oculography; electromyography of anterior tibialis and chin muscles; and electrocardiography.

Respiratory effort was measured by respiratory inductance plethysmography and oxygen saturation by a finger probe connected to a pulse oximeter.

Airflow was measured by means of nasal pressure cannula and thermistor, and snoring was detected by means of a microphone at the suprasternal notch. Children were also monitored on audio/videotape using an infrared camera. Polysomnograms were manually scored by certified technicians according to international guidelines [10–12].

The obstructive apnea–hypopnea index (oAHI) was defined as the number of obstructive apneas and hypopneas per hour of sleep. OSAS was defined as an oAHI ≥ 2/h [13]. OSAS was classified as mild (oAHI between 2 and 5/h), moderate (oAHI 5–10/h), or severe (oAHI ≥ 10/h).

DISE was performed in the operating theater by a single pediatric ear–nose–throat surgeon (A.B). The children were sedated on the operating table with full cardiopulmonary monitoring by a pediatric anesthesiologist using a facial mask with a mixture of sevoflurane and oxygen. When intravenous access was obtained, sevoflurane was stopped and intravenous propofol was administered with a bolus injection of 1–2 mg followed by continuous infusion according to body weight (6–10 mg/kg/h) to obtain the desired level of sedation and to maintain spontaneous breathing. Once a stable respiratory pattern was obtained, a flexible fiberoptic laryngoscope was passed through a swivel adaptor on the mask and introduced into one nostril up to the level of the nasopharynx. No local anesthesia was utilized and care was taken to avoid any pressure from the mask on the patient's face. The examination was performed with the child lying supine and the head in a neutral position. From the nasopharynx, the scope was gently passed toward the oral cavity, the hypopharynx and larynx. At each examination level, the pattern of UA collapse was noted according to a standard protocol and both fixed and dynamic obstructions were noted [14]. Fixed abnormalities are adenoid hypertrophy, tonsillar hypertrophy and tongue enlargement with partial or complete airway compromise.

At the level of the nasopharynx, adenoid hypertrophy was graded as follows: 0, no adenoids; 1, adenoids occupying <50% of the lumen; 2, adenoids occupying 50–75% of the lumen; 3, >75% obstruction

of the nasopharynx by adenoid tissue. Tonsillar obstruction was graded as 0, no tonsils present; 1, <50% collapse of the tonsil; 2, 50–90% collapse of the tonsils; 3, tonsils touch at the midline. Tongue base obstruction was always in anteroposterior direction and scored as absent (0), partial collapse (1), or complete collapse (2).

Dynamic obstructions include hypotonia defined as a circumferential collapse at the oropharyngeal or hypopharyngeal level, anteroposterior palatal collapse or flutter, an anteroposterior collapse of the epiglottis which is sucked against the posterior pharyngeal wall and findings of late-onset laryngomalacia. The latter is characterized by redundant mucosa of the aryepiglottic folds being pulled into the airway and causing UA obstruction during forceful inspiration (type 1 laryngomalacia) [15]. Dynamic collapse was scored as either absent (0) or present (1) for the following parameters: anteroposterior palatal collapse, collapse of the epiglottis, laryngomalacia, hypotonia at oropharyngeal or hypopharyngeal level. The pediatric DISE scoring sheet is shown in Fig. 1.

The flexible fiberoptic laryngoscope was connected to a digital video camera and all endoscopies were digitally recorded and stored for review.

Multi-level obstruction was defined as the presence of one or more UA abnormalities outside the adenotonsillar region. When DISE showed a clinically relevant obstruction at the level of adenoids and/or tonsils, the child was intubated and ventilated at the end of the endoscopic examination and an adenoidectomy or tonsillectomy ± adenoidectomy was performed. Surgery was performed with cold instruments; in the case of tonsillectomy the anterior and posterior tonsillar pillars were sutured with resorbable sutures. If no surgical intervention was required, the child was awakened following the DISE examination and transferred to the recovery room.

All children undergoing a surgical intervention were scheduled for control PSG. Surgical treatment was considered successful with a post-treatment oAHI <5/h. A complete cure was defined as post-treatment oAHI <2/h.

The study was approved by the local Ethics Committee (B30020107827). All parents gave written informed consent.

Statistical analysis was performed with IBM SPSS statistics version 22. Data are reported as median value with lower and upper quartile. Pre-postoperative findings are compared by Wilcoxon signed rank test; correlations between variables were calculated using Pearson's correlation coefficient. Mann–Whitney *U*-test was utilized to compare oAHI between children with obstruction limited to the adenotonsillar region and those with multi-level obstruction. Statistical significance was accepted at *P* < 0.05.

3. Results

Thirty-seven children meeting the inclusion criteria underwent DISE examination during the study period. The study group consisted of 10 girls and 27 boys, aged 4.1 (2.1–6.0) years, BMI 16.20 kg/m² (14.9–15.70), and BMI z-score 0.3 (–0.9 to 0.9). The study population presented with moderate to severe OSAS with oAHI 9.0 (6.1–19.3)/h, mean oxygen saturation (meanSat) of 96.8% (96.0–97.5), and minimum oxygen saturation (minSat) of 89.0% (85.0–93.0).

There were no adverse events during DISE and none of the children needed respiratory support.

A summary of the DISE findings is presented in Figs. 2 and 3. UA obstruction at the level of adenoids and tonsils was found in 33 cases (89%). Twenty-one (56.7%) of the patients had multi-level obstruction. There was no significant difference in oAHI between children with obstruction limited to the adenotonsillar region [8.0 (6.9–16.1)/h] and those with multi-level obstruction [9.4 (5.7–22.4)/h]. Late-onset laryngomalacia was found in two out of the 37 children (5.4%) in combination with adenoid hypertrophy or adenotonsillar

Scoring Sheet – Pediatric DISE

NAME:	DIAGNOSIS:
FIRST NAME:	PREVIOUS SURGERY:
BIRTH DATE:	COMORBIDITY:
EXAMINATION DATE:	
ANESTHESIA:	EXAMINATOR:
Sevoflurane + Propofol	
Propofol only	
Propofol dosage: mg/kg/hr	
FIXED OBSTRUCTION	DYNAMIC COLLAPSE
ADENOIDS	PALATE
0= none	0= no collapse
1=< 50% obstruction	1= collapse present
2= 50% up to 75% obstruction	
3= > 75% obstruction	
TONSILS	EPIGLOTTIS*
0= none	0= no collapse
1=< 50% obstruction	1= collapse present
2= 50% up to 90% obstruction	
3= tonsils touch ad midline	
TONGUE BASE	HYPOTONIA
0= none	0= absent
1= partial obstruction	1= present
2= complete obstruction	
	LARYNGOMALACIA
	0= absent
	1= present
REMARKS: * epiglottis pulled backwards by tonsils or tongue base is not considered as dynamic epiglottis collapse	

Fig. 1. Pediatric drug-induced sedation endoscopy scoring sheet.

hypertrophy. Both presented with a history suggestive for OSAS and there were no complaints of inspiratory stridor.

There was a significant correlation between the Brodsky score for tonsillar hypertrophy and the degree of tonsillar obstruction during DISE ($r = 0.68$, $P = 0.01$) but not between oAHI and the Brodsky score or the degree of tonsillar obstruction during DISE. Thirty-three children underwent a surgical intervention following DISE.

Twenty-eight children with upper airway obstruction at the level of the adenoids and tonsils were treated by ATE. Two children with at least grade 2 obstruction of the adenoids but grade 1 obstruction of the tonsils underwent an adenoidectomy without

tonsillectomy. Two children with grade 1 obstruction of the adenoids and at least grade 2 obstruction of the tonsils during DISE were treated only by tonsillectomy. One of these 33 patients underwent an adenoidectomy and was referred for orthodontic treatment because DISE showed >50% obstruction at the level of the adenoids and complete tongue base obstruction. He did not complete the orthodontic work-up. In the other four patients, a conservative treatment was proposed based upon UA findings during DISE. One patient with grade 1 adenoids and tonsils and epiglottis collapse was treated with montelukast. Two other patients were referred for orthodontic treatment because of complete tongue base

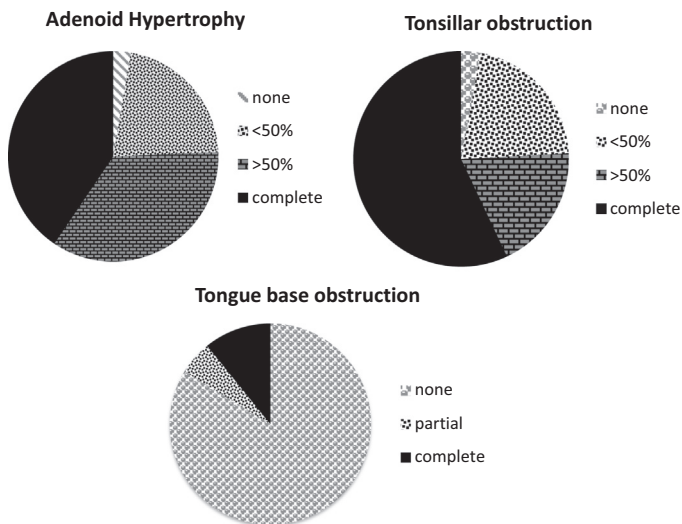


Fig. 2. Drug-induced sedation endoscopy findings for upper airway obstruction at the level of the adenoids, tonsils, and tongue base.

obstruction ($n = 1$) or a collapse of the epiglottis that was relieved by chin lift. Both patients received a removable activator (Twin block activator). One patient was treated by continuous positive airway pressure (CPAP) since he had severe OSAS (oAHI 24.4/h). CPAP was well tolerated but he was referred to the orthodontist to investigate whether orthodontic treatment could be an alternative for CPAP since DISE showed a complete tongue base obstruction. He received a mandibular repositioning device (Somnomed® G2).

DISE-directed treatment options are shown in Fig. 4.

Pre-postoperative PSG data were available for review in 22 patients by November 1, 2013. Among this subgroup, one patient underwent an adenoidectomy, two a tonsillectomy and 19 had undergone an ATE.

Time between surgery and control PSG was 182 (156–259) days. OSAS severity was not significantly different between those children with follow-up PSG data and for those whom control PSG data

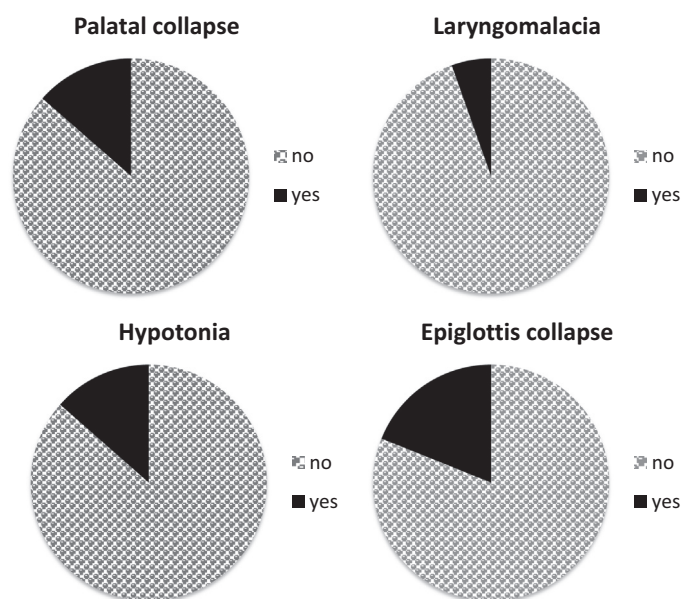


Fig. 3. Drug-induced sedation endoscopy findings for dynamic upper airway collapse at the level of the palate, epiglottis, presence of laryngomalacia and hypotonia.

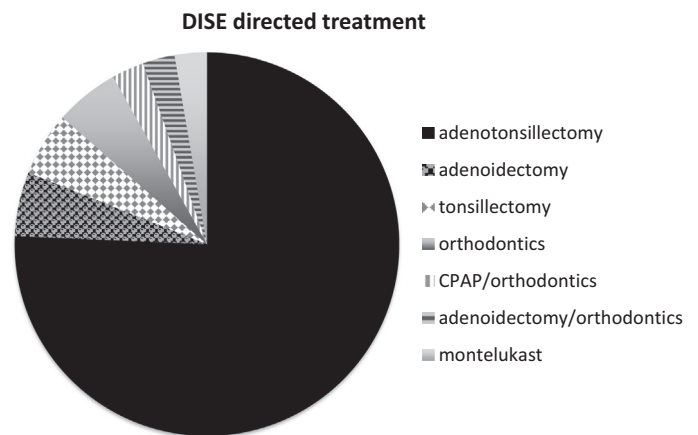


Fig. 4. Drug-induced sedation endoscopy (DISE)-directed treatment options for 37 pediatric obstructive sleep apnea syndrome patients. CPAP, continuous positive airways pressure.

are not available. A significant decrease in oAHI was observed from 8.6/h (6.7–20.7) at baseline to 1.0/h (0.6–2.0) after UA surgery ($P = 0.001$) (Fig. 5) accompanied by a significant improvement in meanSat from 96.7% (96.0–97.5) to 97.5% (97.3–98.0) ($P = 0.003$) and in minSat from 89.0% (85.7–93.0) to 92.0% (89.8–94.0) ($P = 0.03$).

These changes were not accompanied by a significant difference in BMI or sleep architecture. Only two patients were found to have residual OSAS with an oAHI > 5 following surgery, yielding a success rate of 91%. A complete cure was obtained in 17 patients (77%). One patient with moderate residual OSAS was a 4-year-old girl with a history of prematurity. DISE did not indicate collapse or obstruction at levels outside the adenotonsillar region and she underwent ATE. Postoperatively there was residual snoring by history and oAHI increased from 6.8 to 14.3/h. MeanSat did not change (97.6%) but minSat improved from 91% to 93%. The other patient with residual OSAS was a 7-month-old boy in whom DISE showed hypotonia in combination with adenotonsillar hypertrophy. He had a common cold at the time of the postoperative PSG and oAHI increased from 19.7 to 30.9/h with an improvement in meanSat (from 97.3% to 98%) and minSat (from 86% to 91%). In the patient treated with montelukast, a control PSG 224 days from baseline PSG showed a decrease in oAHI from 17 to 1.8/h. One of the patients treated with an activator had a complete cure at the end of the orthodontic treatment.

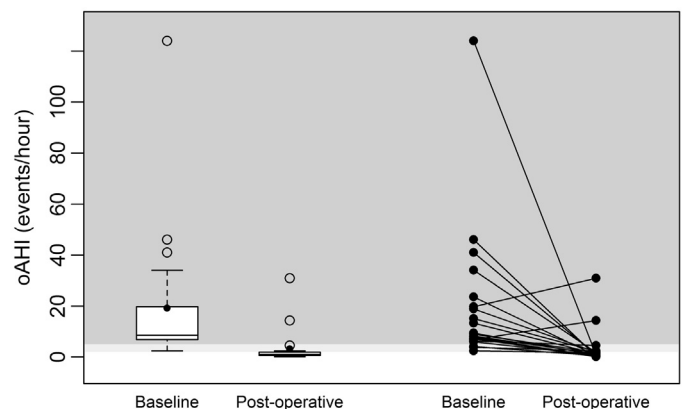


Fig. 5. Pre-postoperative obstructive apnea-hypopnea index (oAHI) in 22 patients. Left-hand plot shows median values with upper and lower quartile. Right-hand plot shows values for individual cases.

4. Discussion

We report data on a homogeneous group of OSAS children without a history of previous upper airway surgery and without syndromic comorbidity or craniofacial abnormalities. In addition, we provide outcome data on DISE-directed surgery.

Truong et al. [8] reported DISE-directed treatment outcomes in 35 children undergoing ATE but 28.2% of these subjects were considered hypotonic or had syndromic comorbidities.

Therefore, our paper may be considered the first report on DISE findings and treatment outcomes in a carefully selected population of surgically naive OSAS children without syndromic comorbidities or craniofacial abnormalities.

DISE allowed for an individualized treatment proposal and 11% of children were not considered surgical candidates. The therapeutic improvement in this prospective cohort of children undergoing DISE-directed UA surgery suggests an improved outcome compared to available data in literature.

Pediatric DISE was first described in 1990 by Croft et al. [16] and later by Myatt and Beckenham in children with complex UA disorders [6]. Truong et al. reported outcome data for DISE-directed treatment in 39 OSAS patients who had never undergone UA surgery, and they found a significant improvement in AHI from 13.8 ± 15.9 to $8.0 \pm 8.0/h$ [8]. However, 28.2% of these patients were considered hypotonic or had a syndromic comorbidity. Fishman et al. compared DISE with awake flexible endoscopy in children with persistent OSAS following ATE and concluded that DISE is a reliable and valuable tool for identifying sites and structures contributing to UA obstruction in children with persistent OSAS [7].

Success rate of ATE in the literature varies depending on the study population and associated obesity. Although ATE has a 97% success rate by parental reports, a recent meta-analysis estimated a 66.2% (confidence interval, 54.5–76.3) success rate for ATE in normal children [17]. Success was defined as $AHI < 5$ and it should be acknowledged that $\geq 75\%$ of children in this analysis were obese or had severe OSAS. Limiting the analysis to ‘uncomplicated’ patients, the cure rate was 73.8%.

In the present study, surgical candidates were selected on the basis of PSG and UA findings during DISE and the outcomes compare favorably to those in the literature. Only 9% of our surgically treated patients for whom pre-postoperative data were available had residual OSAS and 77% achieved a complete cure. Upon analyzing the clinical records of the two patients with persistent OSAS despite adenotonsillar obstruction during DISE, other factors such as history of prematurity, findings of hypotonia and presence of a common cold at the time of the control PSG might have contributed to the poor outcome in these cases.

In adults, several authors emphasized the importance of a standardized protocol for reporting DISE findings [18,19]. The VOTE classification was developed to allow for a uniform assessment of the degree of UA narrowing at four levels of the UA (velum, oropharynx/lateral walls, tongue base and epiglottis) [19]. Ulualp and Szmuk utilized the VOTE classification in pediatric DISE [20]. We believe, however, that adult findings cannot merely be translated to the pediatric airway. The VOTE classification does not include a scoring for adenoid hypertrophy/obstruction or for laryngomalacia, two findings that are highly relevant in pediatric OSAS. Durr et al. used the VOTE classification in children with persistent OSAS but included three items: inferior turbinate hypertrophy, adenoid regrowth, and laryngomalacia [21]. Truong et al. [8] and Lin and Koltai [22] proposed a qualitative description of DISE findings at four levels: velopharynx (including adenoids and soft palate), oropharynx (tonsils and circumferential collapse of the lateral pharyngeal walls), hypopharynx (lingual tonsillar hypertrophy, tongue base obstruction, and circumferential collapse), and supraglottis (occult laryngomalacia). Although this classification system also accounts

for UA signs of hypotonia, it does not address the degree of UA obstruction (partial/complete) at a certain airway level. Goldberg et al. emphasized the importance of dynamic UA collapse in pediatric OSAS [14]. Dynamic abnormalities were found to be frequent among hypotonic children and the authors suggested that dynamic defects are less likely to respond to surgical intervention.

We developed a comprehensive and standardized scoring protocol for pediatric DISE, taking into account both structural and dynamic UA abnormalities in combination with a quantification of the degree of UA obstruction. Our scoring system allowed us to classify DISE findings in all pediatric OSAS patients (including those with previous UA surgery or comorbidities not included in this paper).

If we define multi-level obstruction as the presence of one or more abnormal UA findings at levels outside the adenotonsillar region, 21 out of 37 patients (56.7%) had multi-level obstruction. This finding is in line with previous reports in the literature [11,18,23]. Ulualp et al. [20] concluded that children with mild OSA more often had single-site obstruction whereas those with moderate to severe OSAS had obstruction at multiple sites of the upper airway. In our data, no significant difference was found in OSAS severity between those with multi-level obstruction and children with obstruction limited to the adenotonsillar region.

The role of DISE in the routine work-up of pediatric OSAS patients is not yet established. Few authors have reported on the systematic use of endoscopic airway evaluation in children undergoing UA surgery for OSAS [15,24].

Second, most previous papers focused on selected children such as those with complex UA obstruction or persistent OSAS following ATE [6–8,21].

The 9% failure rate after DISE-directed UA surgery in our present study supports the value of DISE in the assessment of children with OSAS. Further studies from other independent centers are required to support this concept. In addition, studies in children with complex OSAS such as those with obesity or Down syndrome are ongoing and may provide more compelling evidence regarding the clinical value of pediatric DISE. Our findings are in line with previous reports that DISE is a reliable tool for UA evaluation in pediatric OSAS patients [21,22].

We found a 5.4% prevalence of late-onset laryngomalacia. This is comparable with the 3.9% prevalence rate reported by Thevasagayam et al. [24]. Late-onset laryngomalacia is clinically distinct from congenital laryngomalacia [15]. Children with late-onset laryngomalacia typically have a type 1 laryngomalacia and do not present with stridor but with typical OSAS symptoms [15].

Although the clinical evaluation of tonsillar hypertrophy by the Brodsky score correlated strongly with the degree of tonsillar obstruction during DISE, neither parameter correlated significantly with OSAS severity in terms of oAHI values. A similar finding was reported by Van Holsbeke et al. [10]. These authors found that functional parameters of UA patency derived from computed tomography correlated with OSAS severity, whereas clinical parameters of UA obstruction did not. In the future, it would be interesting to compare data from computed tomography analyzed by computational fluid dynamics with DISE findings.

Our study has several limitations. Sedation was achieved by inhalation of sevoflurane followed by intravenous administration of propofol. Propofol was the only drug used in those patients for whom venous access could be obtained while they were fully awake. Propofol is the first-choice drug for DISE in adults. The drug is metabolized quickly and has a low incidence of side-effects such as nausea, vomiting, and headache [11]. It has been utilized in several previous studies performed in children [7,8,14,15,21], where it produced reliable findings [8,21]. Some authors criticized the use of propofol for DISE which caused excessive hypotonia and muscle relaxation with alteration of airway dynamics, and instead advocated the use dexmedetomidine and ketamine [22]. Dexmedetomidine

would induce less muscle relaxation and produce a more sustained respiratory effort. However, this drug has other side-effects and may result in transient changes in blood pressure and heart rate [23,25]. In Europe, dexmedetomidine is licensed only for prolonged sedation of adult patients in an intensive care unit. The use of this drug for DISE in children in our hospital would imply off-label use and would not be justified for this indication.

Second, control PSG data were not available for all surgically treated children. However, there was no significant difference in OSAS severity in those with or without control PSG data, so we do not believe that there is a selection bias in the reported outcomes. A third limitation is that we do not yet have outcome data on a larger group of children for whom a non-surgical and non-CPAP treatment was proposed based upon DISE findings. These data are required to validate further the use of DISE in pediatric OSAS patients and to justify a non-surgical treatment (medication or orthodontics) even in those with moderate to severe disease.

At present, we propose DISE as a routine examination in all pediatric OSAS patients prior to UA surgery and irrespective of any previous UA surgery or comorbidity. Treatment decisions based upon the anatomical findings observed during DISE may improve surgical treatment outcomes. It should also be emphasized that in some patients considered as surgical candidates based upon clinical examination and PSG data, DISE findings prompted us to propose a conservative treatment instead of surgery or a combination of surgery and orthodontic treatment.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <http://dx.doi.org/10.1016/j.sleep.2014.06.016>.

Acknowledgments

The authors would like to thank Miss Kristien Wouters, statistician at the University Hospital Antwerp for her help with the figures and data management. We are also indebted to Prof. Dr. O. Vanderveken, Department of Otorhinolaryngology, Head and Neck Surgery, University Hospital Antwerp, for his critical review of the manuscript.

References

- [1] Lumeng JC, Chervin RD. Epidemiology of pediatric obstructive sleep apnea. *Proc Am Thorac Soc* 2008;5:242–52.
- [2] Ye J, Liu H, Zhang GH, Li P, Yang QT, Liu X, et al. Outcome of adenotonsillectomy for obstructive sleep apnea syndrome in children. *Ann Otol Rhinol Laryngol* 2010;119:506–13.
- [3] Mitchell RB, Kelly J. Outcome of adenotonsillectomy for obstructive sleep apnea in obese and normal-weight children. *Otolaryngol Head Neck Surg* 2007;137:43–8.
- [4] Vanderveken OM. Drug-induced sleep endoscopy (DISE) for non-CPAP treatment selection in patients with sleep-disordered breathing. *Sleep Breath* 2013;17:13–14.
- [5] Koutsourelakis I, Safiruddin F, Ravesloot M, Zakynthinos S, de Vries N. Surgery for obstructive sleep apnea: sleep endoscopy determinants of outcome. *Laryngoscope* 2012;122:2587–91.
- [6] Myatt HM, Beckenham EJ. The use of diagnostic sleep nasendoscopy in the management of children with complex upper airway obstruction. *Clin Otolaryngol* 2000;25:200–8.
- [7] Fishman G, Zemel M, DeRowe A, Sadot E, Sivan Y, Koltai PJ. Fiber-optic sleep endoscopy in children with persistent obstructive sleep apnea: inter-observer correlation and comparison with awake endoscopy. *Int J Pediatr Otorhinolaryngol* 2013;77:752–5.
- [8] Truong MT, Woo VG, Koltai PJ. Sleep endoscopy as a diagnostic tool in pediatric obstructive sleep apnea. *Int J Pediatr Otorhinolaryngol* 2012;76:722–7.
- [9] Brodsky L. Modern assessment of tonsils and adenoids. *Pediatr Clin North Am* 1989;36:1551–69.
- [10] Van Holsbeke C, Vos W, Van Hoorenbeeck K, Boudewyns A, Salgado R, Verdonck PR, et al. Functional respiratory imaging as a tool to assess upper airway patency in children with obstructive sleep apnea. *Sleep Med* 2013;14:433–9.
- [11] Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. *J Clin Sleep Med* 2012;8:597–619.
- [12] Iber C, Ancoli-Israel S, Chesson AL, Quan SF for the American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester, IL: AASM; 2007.
- [13] Marcus CL, Moore RH, Rosen CL, Giordani B, Garetz SL, Taylor HG, et al. A randomized trial of adenotonsillectomy for childhood sleep apnea. *N Engl J Med* 2013;368:2366–76.
- [14] Goldberg S, Shatz A, Picard E, Wexler I, Schwartz S, Swed E, et al. Endoscopic findings in children with obstructive sleep apnea: effects of age and hypotonia. *Pediatr Pulmonol* 2005;40:205–10.
- [15] Revell SM, Clark WD. Late-onset laryngomalacia: a cause of pediatric obstructive sleep apnea. *Int J Pediatr Otorhinolaryngol* 2011;75:231–8.
- [16] Croft CB, Thomson HG, Samuels MP, Southall DP. Endoscopic evaluation and treatment of sleep-associated upper airway obstruction in infants and young children. *Clin Otolaryngol* 1990;15:209–16.
- [17] Friedman M, Wilson M, Lin HC, Chang HW. Updated systematic review of tonsillectomy and adenoidectomy for treatment of pediatric obstructive sleep apnea/hypopnea syndrome. *Otolaryngol Head Neck Surg* 2009;140:800–8.
- [18] Rodriguez-Bruno K, Goldberg AN, McCulloch CE, Kezirian EJ. Test–retest reliability of drug-induced sleep endoscopy. *Otolaryngol Head Neck Surg* 2009;140:646–51.
- [19] Kezirian EJ, Hohenhorst W, de Vries N. Drug-induced sleep endoscopy: the VOTE classification. *Eur Arch Otorhinolaryngol* 2011;268:1233–6.
- [20] Ulualp SO, Szmuk P. Drug-induced sleep endoscopy for upper airway evaluation in children with obstructive sleep apnea. *Laryngoscope* 2013;123:292–7.
- [21] Durr ML, Meyer AK, Kezirian EJ, Rosbe KW. Drug-induced sleep endoscopy in persistent pediatric sleep-disordered breathing after adenotonsillectomy. *Arch Otolaryngol Head Neck Surg* 2012;138:638–43.
- [22] Lin AC, Koltai PJ. Sleep endoscopy in the evaluation of pediatric obstructive sleep apnea. *Int J Pediatr* 2012;2012:576719.
- [23] Jooste EH, Muhly WT, Ibinson JW, Suresh T, Damian D, Phadke A, et al. Acute hemodynamic changes after rapid intravenous bolus dosing of dexmedetomidine in pediatric heart transplant patients undergoing routine cardiac catheterization. *Anesth Analg* 2010;111:1490–6.
- [24] Thevasagayam M, Rodger K, Cave D, Witmans M, El-Hakim H. Prevalence of laryngomalacia in children presenting with sleep-disordered breathing. *Laryngoscope* 2010;120:1662–6.
- [25] Mason KP, Zgleszewski SE, Prescilla R, Fontaine PJ, Zurakowski D. Hemodynamic effects of dexmedetomidine sedation for CT imaging studies. *Paediatr Anaesth* 2008;18:393–402.